



Determinants of flow-mediated outward remodeling in female rodents: respective roles of age, estrogens, and timing

Submitted by Emmanuel Lemoine on Tue, 02/24/2015 - 15:26

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| Titre | Determinants of flow-mediated outward remodeling in female rodents: respective roles of age, estrogens, and timing |
| Type de publication | Article de revue |
| Auteur | Tarhouni, K. [1], Guihot, Anne-Laure [2], Vessieres, Emilie [3], Toutain, Bertrand [4], Procaccio, Vincent [5], Grimaud, Linda [6], Loufrani, Laurent [7], Lenfant, F. [8], Arnal, Jean-François [9], Henrion, Daniel [10] |
| Editeur | American Heart Association |
| Type | Article scientifique dans une revue à comité de lecture |
| Année | 2014 |
| Langue | Anglais |
| Date | 2014 |
| Numéro | 6 |
| Pagination | 1281 - 9 |
| Volume | 34 |
| Titre de la revue | Arteriosclerosis, Thrombosis, and Vascular Biology |
| ISSN | 1524-4636 |
| Mots-clés | Aging [11], Animals [12], Endothelium, Vascular/physiology [13], Estradiol/blood/physiology [14], Estrogen Receptor alpha/analysis [15], Extracellular Signal-Regulated MAP Kinases/physiology [16], Female [17], Mesenteric Arteries/pathology/physiology [18], Nitric Oxide Synthase Type III/analysis [19], Ovariectomy [20], Rats [21], Rats, Wistar [22], Regional Blood Flow [23], Stress, Mechanical [24], Superoxide Dismutase/metabolism [25], Time Factors [26], Vascular Resistance [27], Vasodilation [28] |

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| Résumé en anglais | <p>OBJECTIVE: Flow (shear stress)-mediated outward remodeling (FMR) of resistance arteries is a key adaptive process allowing collateral growth after arterial occlusion but declining with age. 17-beta-estradiol (E2) has a key role in this process through activation of estrogen receptor alpha (ERalpha). Thus, we investigated the impact of age and timing for estrogen efficacy on FMR. APPROACH AND RESULTS: Female rats, 3 to 18 months old, were submitted to surgery to increase blood flow locally in 1 mesenteric artery in vivo. High-flow and normal-flow arteries were collected 2 weeks later for in vitro analysis. Diameter increased by 27% in high-flow arteries compared with normal-flow arteries in 3-month-old rats. The amplitude of remodeling declined with age (12% in 18-month-old rats) in parallel with E2 blood level and E2 substitution failed restoring remodeling in 18-month-old rats. Ovariectomy of 3-, 9-, and 12-month-old rats abolished FMR, which was restored by immediate E2 replacement. Nevertheless, this effect of E2 was absent 9 months after ovariectomy. In this latter group, ERalpha and endothelial nitric oxide synthase expression were reduced by half compared with age-matched rats recently ovariectomized. FMR did not occur in ERalpha(-/-) mice, whereas it was decreased by 50% in ERalpha(+/-) mice, emphasizing the importance of gene dosage in high-flow remodeling. CONCLUSIONS: E2 deprivation, rather than age, leads to decline in FMR, which can be prevented by early exogenous E2. However, delayed E2 replacement was ineffective on FMR, underlining the importance of timing of this estrogen action.</p> |
| URL de la notice | http://okina.univ-angers.fr/publications/ua8211 [29] |
| DOI | 10.1161/ATVBAHA.114.303404 [30] |
| Lien vers le document | http://dx.doi.org/10.1161/ATVBAHA.114.303404 [30] |
| Titre abrégé | Arterioscler Thromb Vasc Biol |

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- [30] <http://dx.doi.org/10.1161/ATVBAHA.114.303404>

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